

A motion area in human visual cortex

(kinetic boundaries/orientation/discrimination/positron emission tomography)

GUY A. ORBAN*, PATRICK DUPONT†, BART DE BRUYN*, RUFIN VOGELS*, RIK VANDENBERGHE*,
AND LUC MORTELMANS†

*Laboratorium voor Neuro- en Psychofysiologie, Katholieke Universiteit te Leuven, Faculteit der Geneeskunde, Campus Gasthuisberg, B-3000 Leuven, Belgium; and †Positron Emission Tomography Center, Department of Nuclear Medicine, Universitair Ziekenhuis Gasthuisberg, B-3000 Leuven, Belgium

Communicated by James M. Sprague, University of Pennsylvania Medical Center, Philadelphia, PA, October 10, 1994 (received for review July 1, 1994)

ABSTRACT We have localized an area in the human brain involved in the processing of contours defined by motion differences (kinetic contours) by comparing with positron emission tomography the regional cerebral blood flow in tasks performed with kinetic and luminance-defined gratings. These tasks included passive viewing, counting the total number of grating stimuli, and counting the number of gratings of a given orientation. Comparison between the counting tasks and passive viewing with a given type of contour revealed a set of active areas that were similar for both luminance-defined and kinetic contours. Comparisons between these two types of contours revealed a single focus in the right hemisphere that did not overlap with the many regions activated by uniform motion. In particular this “kinetic focus” was clearly separated from the area previously defined as the human homologue of V5/middle temporal. Activity in this kinetic focus was stronger when orientation had to be processed than in the other two tasks. These results and control experiments with uniformly moving random dot patterns suggest the existence of an area in the human visual system that is activated much more by kinetic contours than by luminance contours or uniformly moving random dots. Up to now, such an area has not been described in the monkey visual system.

Motion is an important visual attribute, and the analysis of retinal image movement can subserve many functions (1) in addition to motion perception. One such function is segmentation and extraction of kinetic contours. Segmentation of a figure from the background is an early visual process essential for object recognition. Motion segmentation is the only manner to detect distant objects camouflaged by a background with similar luminance and color spatial distributions.

Recent studies of the primate visual cortex have revealed some of the areas involved in the processing of kinetic contours. The middle temporal (MT) area/V5 (2, 3) has been identified as playing a critical role in the analysis of retinal image motion. It contains a very large fraction of direction-selective and speed-tuned cells (4–6); its destruction leads to deficits in judgments of speed and direction of motion, in initiation of smooth pursuit, and in the perception of kinetic shapes (7–11). Although the lesion data suggest that MT is involved in the processing of kinetic contours, MT cells are not selective for the orientation of kinetic contours (12). On the other hand, it has been shown that the shape selectivity of inferotemporal cells is cue invariant: shape preference was similar for kinetic and luminance-defined stimuli (13). This probably reflects an invariance present at earlier levels, because a number of V2 and V4 cells have been reported to be

similarly tuned for orientation of kinetic and luminance-defined contours (14, 15).

From these results, we have derived the following hypothesis (16): kinetic boundaries require an additional motion preprocessing operation compared to luminance-defined contours, and this preprocessing takes place in area MT. The preprocessed MT signals are then fed back into the ventral stream running from V1 through V2 and V4 to the inferotemporal cortex (17), and from that reentry point (either V1 or V2) processing is similar for either contour. From this hypothesis we made two predictions: first, compared with a control task, such as passive viewing, tasks requiring processing of orientation should activate similar structures for luminance- or motion-defined stimuli, and, second, comparison of identical tasks for motion- or luminance-defined stimuli should reveal the motion preprocessing stage and thus MT. These predictions were tested in the human visual system using the positron emission tomography activation techniques (18). The first prediction was confirmed, but testing the second prediction revealed a motion area other than that identified as the human homologue of MT/V5 (19, 20).

METHODS

Regional cerebral blood flow (rCBF) was measured with $H_2^{15}O$ injected i.v. Stimuli were rectangular gratings presented for 200 msec by an Atari computer at 70 Hz. Stripe width was 0.66° , diameter was 3° , and mean luminance was 47.5 candelas (cd/m^2). Stimuli were centered on the fixation target. They were generated by modulation of random textured patterns (50% white and dark pixels of 1 arc min), and the stripes differed either in luminance (Lum; 28.8 versus 66.1 cd/m^2) or in motion direction (Kin; leftward versus rightward). In kinetic gratings, pixels moved at $4^\circ/\text{sec}$. Orientation could be manipulated independently of the defining cue. Trials involved presentation at 3 Hz of strings of 9, 10, or 11 stimuli; 4, 5, or 6 of them were vertical, and the others were slightly oblique (orientation difference, 5° for Lum and 13° for Kin). The subjects were asked (i) to fixate with no decision about the stimuli (Passive), (ii) to count the total number of gratings (Total Count) and press the right or left key depending on whether or not the number was 10, and (iii) to count the number of oblique gratings (Orientation Count) and press the right or left key depending on whether or not this number was 5. Combination with the two types of gratings, luminance-defined (Lum) and motion-defined (Kin), yielded a total of six conditions. The order of testing of the conditions was randomized across subjects. Subjects had to report within 1.25 sec after the end of the last stimulus in the string. Intertrial interval was 2.5 sec. Subjects gave their informed consent and were trained in two sessions prior to the positron emission tomography scanning.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. §1734 solely to indicate this fact.

Abbreviations: AC, anterior commissure; PC, posterior commissure; MT, middle temporal; rCBF, regional cerebral blood flow.

Brain activity was monitored as relative changes in local blood flow using the $H_2^{15}O$ method (CTI scanner 931/08/12, Knoxville, TN). The head of each subject was immobilized with a foam headholder. A transmission scan was obtained to correct for attenuation. Each subject underwent six emission scans at least 15 min apart. Subjects received an i.v. injection of 50 mCi (1 Ci = 37 GBq) of $H_2^{15}O$ in 12 sec at the beginning of each task, lasting 2 min. The emission scan (40 sec) started when radioactivity reached the brain (in general, 30 sec after injection). Radioactivity was measured in 15 planes, parallel to the inferior orbitomeatal line and spaced 6.75 mm apart. Fixation was controlled by an electro-oculogram. Seven male and seven female subjects performed the tasks well: percent correct was on average 85% for the Orientation Count tasks and 92% for the Total Count tasks and was similar for the two types of contours. The differences in rCBF between conditions were analyzed with SPM software (21), and foci were defined by significance levels of $P < 0.001$ in at least two horizontal sections made in stereotactic coordinates (22, 23). By requiring significant foci to extend over at least two horizontal sections (smoothing in z axis: full width at half-maximum 12 mm), we reduce the chance of false positives, which are not completely eliminated by setting the threshold for the z score at 3.1 ($P < 0.001$). Two types of subtractions were made: between different tasks for a given type of contour and between types of contours for a given task.

RESULTS

Comparison of the counting tasks with the passive tasks revealed activations that were similar for the two counting

tasks and for the two types of stimuli. Figs. 1 and 2 show that the active areas revealed by the Orientation Count – Passive subtractions are similar for the two types of contours. These activations include right area 18 on the medial side, the thalamus region, the cerebellum, and right parietal cortex. Table 1 illustrates that this also holds for the two types of tasks performed with the same type of stimulus, in this case luminance-defined stimuli. The thalamic, medial area 18, and most cerebellar activations are common to the two tasks. This similarity between Total Count and Orientation Count is in agreement with an earlier studies (24–26) showing little difference in the occipital cortex between detection and orientation identification tasks.

The four counting tasks activated a right medial area 18 focus that was observed in all three previous studies on orientation discrimination (24–26). This medial area 18 activation was equally strong whether orientation had to be processed or not (Fig. 3A). This suggests that this activation might reflect signaling the presence of a contour as much as signaling its orientation. This medial area 18 focus is located more anteriorly and below the activations observed by Corbetta *et al.* (27) in the calcarine region when subtracting passive from active conditions. This is not surprising since the stimulus of Corbetta *et al.* was 32° in diameter and ours was only 3° . According to recent data (28), activation of area 17 by stimuli at 1.5° eccentricity extend only 10 mm into the calcarine from the occipital pole, while 15° eccentricity corresponds to 25 mm distance from the representation of the fixation point. Thus while the foci of Corbetta *et al.* are likely to represent activations of area 17 and are listed as such in their report, the medial area 18 focus is located too far anterior to belong to

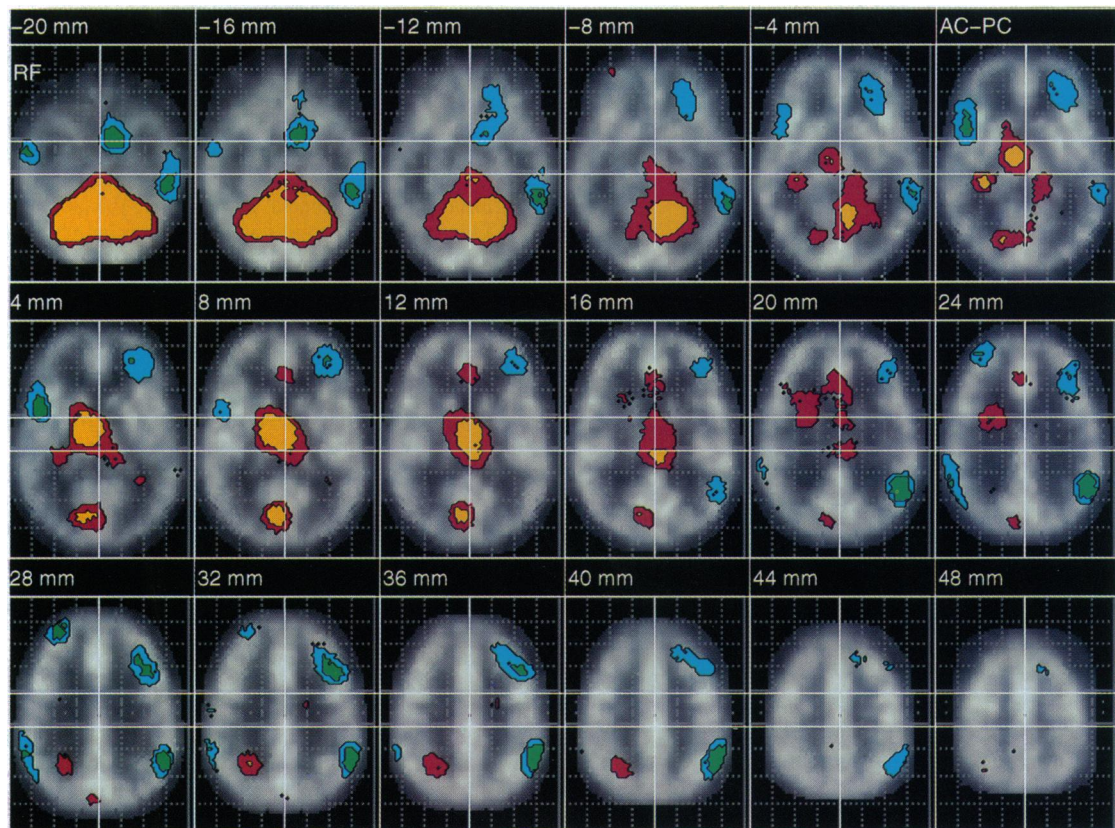


FIG. 1. Regions differing significantly between the experimental condition (Orientation Count) and the control conditions (Passive) for luminance-defined gratings: yellow and red indicate pixels with increased rCBF in the experimental condition significant at $P < 0.001$ and 0.01 levels, respectively; green and blue indicate pixels with decreased rCBF significant at $P < 0.001$ and 0.01 levels, respectively. These regions are superimposed on horizontal sections, from -20 below to 48 mm above the anterior commissure (AC)–posterior commissure (PC) line, through the average rCBF scans from all conditions and subjects to show the anatomical brain features. RF in the lowest section indicates right frontal; in these sections, right hemisphere is on the left of the section.

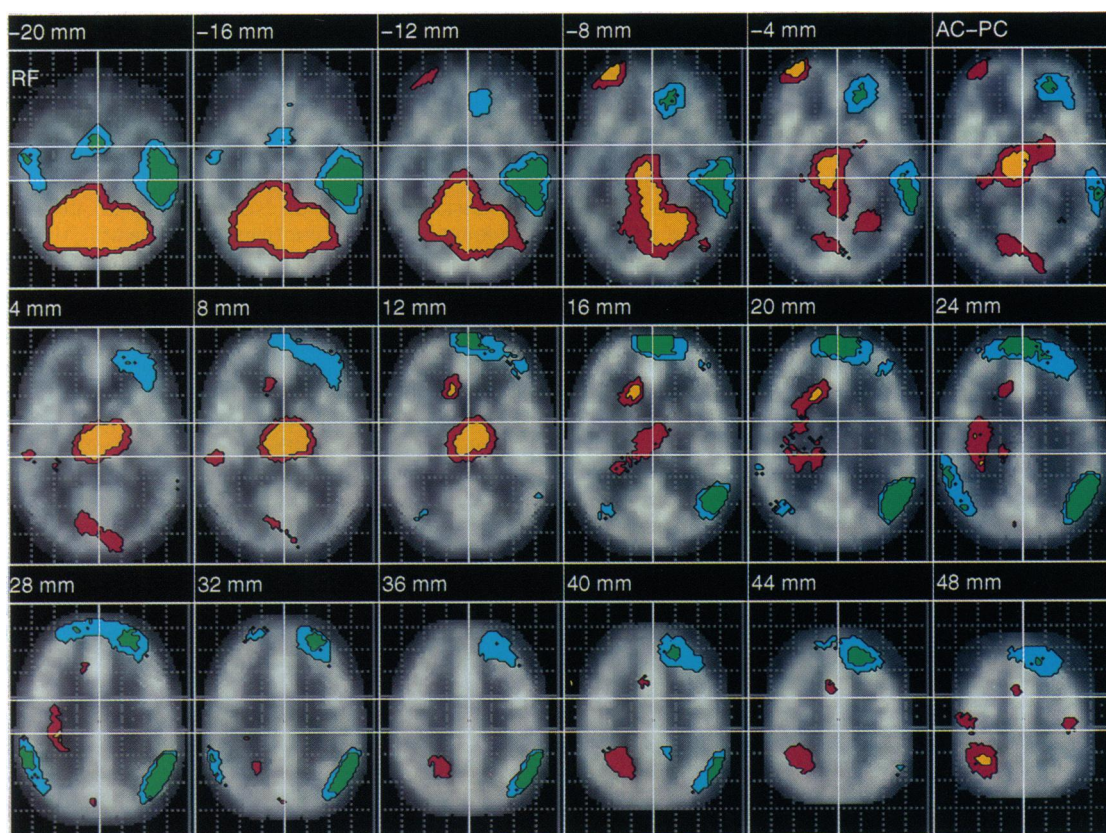


FIG. 2. Regions differing significantly between the Orientation Count and the Passive condition for kinetic gratings. Conventions are as given in Fig. 1.

area 17. The activation in the thalamus region has been linked to the use of internal standards (25). The present data suggest that these activations could also reflect a form of working memory with storage of events. Finally the activation of cerebellum is in agreement with all our previous studies, which

Table 1. Activation sites for tasks using luminance-defined stimuli

Coordinates			z score	
x	y	z	Orientation count	Total count
<i>Cerebellum</i>				
-16	-54	-22	5.53	5.22
-4	-58	-19	4.70	4.37
15	-56	-15	4.84	4.88
-10	-46	-14	3.45	4.59
-2	-44	-24	4.72	4.88
17	-66	-18	4.41	NS
5	-65	-12	4.93	NS
<i>Thalamus</i>				
-6	-12	6	4.42	4.31
6	-28	14	3.27	NS
-5	-25	10	3.33	NS
<i>Hippocampus</i>				
-30	-33	-2	3.38	NS
<i>Cortex</i>				
-9	-73	7 [†]	3.54	3.23
-23	-56	34	3.20	NS

Coordinates are distance in mm from AC-PC line (x, z) and from the AC (y) in Talairach space. Negative values indicate right (x), low (z), and posterior (y) locations. z scores are given for the comparisons Orientation Count - Passive and Total Count - Passive viewing. NS, not significant.

*Distinction between hippocampus and tail of the caudate is difficult.

[†]Medial area 18 focus (see Fig. 3).

have always revealed cerebellar activations when subjects were required to perform discriminations as fast as possible. Con-

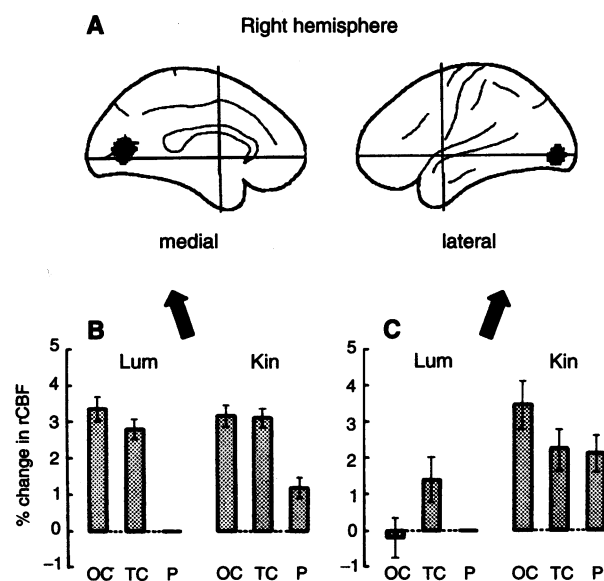


FIG. 3. Location of the medial and lateral area 18 foci in the right hemisphere (A) and percent change in rCBF in the five conditions compared to the Passive/Lum condition for the medial area 18 focus ($x = -9, y = -73, z = 7$) (B) and the lateral area 18 kinetic focus ($x = -25, y = -88, z = -1$) (C), where average coordinates are distances from the AC-PC (x, z) and from the AC (y). The average coordinates were obtained by averaging the local optima in the different sections and subtractions in which the z score exceeded the 3.1 threshold. Error bars indicate SEMs across the subjects. P, passive; TC, total count; OC, orientation count.

trary to previous studies, however, there is no transformation of every visual stimulus into a motor command, but rather a transformation of a visual stimulus into an abstract, nonvisual representation of an event.

There are a number of differences between the two subtractions shown in Figs. 1 and 2. For the kinetic contours, the thalamic activation is more bilateral than for the luminance-defined contours; also the right parietal activations are somewhat stronger in the kinetic than in the luminance cases. Finally, there was a right frontal activation and a clearer anterior cingulate activation in the kinetic case than in the luminance case. There are also a number of sites activated in Orientation Count that do not reach significance in Total Count (Table 1).

When Lum was subtracted from Kin for each of the three types of tasks, only the subtraction of the two Orientation Count conditions revealed a significant focus (Fig. 4). This focus was located on the lateral side of the right occipital cortex (area 18) at levels -4 to 4 mm (Fig. 4B). A difference significant at the $P < 0.01$ level was also obtained for this "kinetic focus" of lateral area 18 in the comparison of the two passive tasks (Fig. 4A). For the kinetic focus we calculated the change in rCBF with respect to the Passive/Lum condition for each of the five other conditions. There was a larger increase in the three kinetic conditions than in the two other conditions

(Fig. 3B). The increases in the Passive/Kin and Total Count/Kin conditions were close to 2% compared to 3.25% in the Orientation Count/Kin condition (Fig. 3B). The kinetic focus is at least 20 mm more posterior than the proposed human homologue of MT/V5 (19, 20). In fact the kinetic focus in lateral area 18 is just posterior to the focus on the occipital convexity activated in a simultaneous orientation discrimination task (24). Control experiments with uniformly moving random dot patterns confirmed that the MT/V5 focus does not overlap with the kinetic focus and that this kinetic focus is less responsive to uniform motion than the surrounding areas (Fig. 4C). Furthermore, comparison of Fig. 4B and C shows that the activation of the MT/V5 homologue was bilateral while the kinetic focus was lateralized in the right hemisphere. The Kin – Lum subtractions revealed no significant differences in the region corresponding to the human homologue of MT, although comparison of the passive tasks yielded a nonsignificant difference as that level in the right hemisphere (Fig. 4A).

DISCUSSION

Our results suggest that there are at least two types of motion areas in the human brain: areas activated by uniform motion, which include the region referred to as the human homologue of MT/V5 (19, 20), and an area involved in the processing of

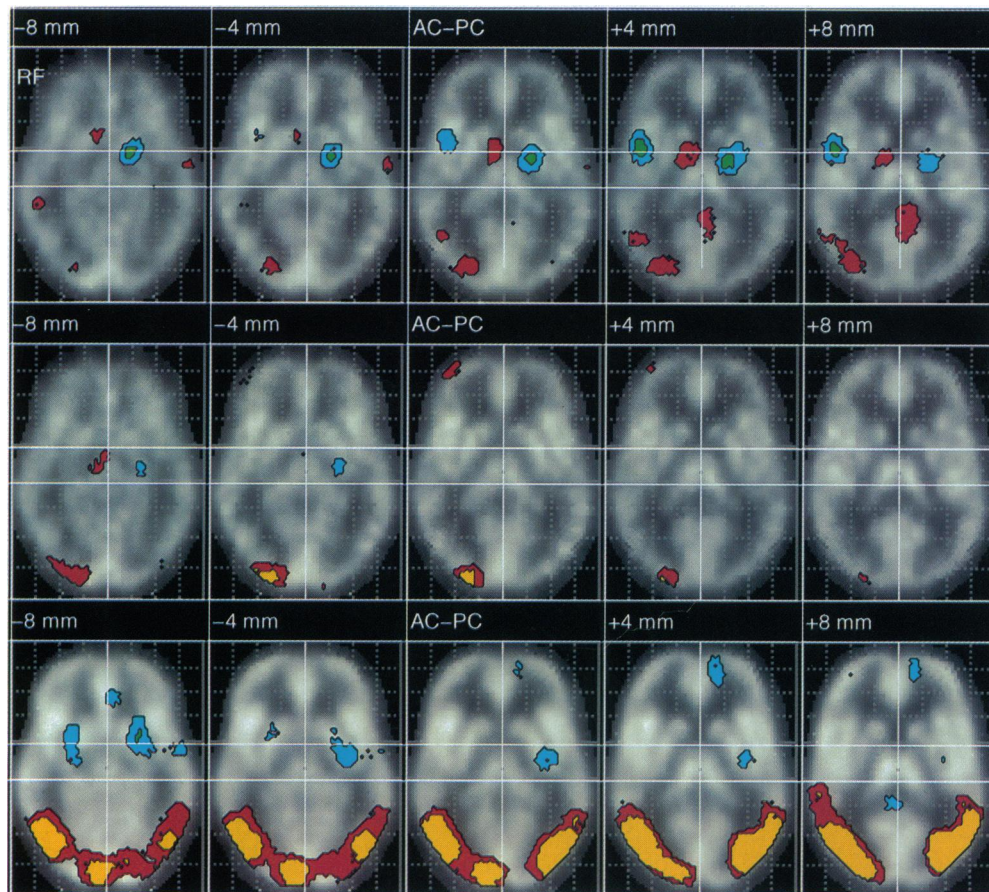


FIG. 4. Regions with significant difference in the Passive/Kin compared to Passive/Lum (Top), in the Orientation Count/Kin compared to Orientation Count/Lum (Middle), and in uniform moving random dot patterns compared to stationary random dot patterns (Bottom). Only sections -8 to $+8$ mm are shown. Other conventions are as given in Fig. 1. (Bottom) Data from 14 different subjects from Dupont *et al.* (29). Subjects were only required to keep fixation; the stimulus was a 3° random dot field (mean luminance = 3.8 cd/m^2 , dots of 2 arc min size, $28.3 \text{ dots per deg}^2$), with dots moving at $4^\circ/\text{sec}$ in one of eight directions (45° steps) and changing direction every 428 msec. Because this random dot pattern has proportionally more energy at low spatial frequencies, it is more powerful than the random textured stimulus used to define the kinetic gratings: a coarse random dot pattern drives macaque MT cells a factor 1.8 better than a fine random textured pattern (V. L. Marcar, S. E. Raiguel, D. Xiao, H. Maes, and G.A.O., unpublished results). It is also more powerful than that of Watson *et al.* (20), because of the larger dot density (24, 25). Calculating the percent increase rCBF in the moving-stationary random dot pattern comparison of C, we obtained 3.53% increase in the kinetic focus (A18 laterally) compared to 5.59% and 5.60% in right and left MT/V5 analogues (average coordinates: $x = -44$, $y = -67$, $z = 3$ and $x = +40$, $y = -70$, $z = 4$, respectively).

kinetic contours. This distinction is in agreement with psychophysical observations of Chang and Julesz (30). It is also in agreement with patient data. Vaina (31) reported a double dissociation between kinetic form perception impaired after right occipitotemporal lesions and speed discrimination impaired after right occipitoparietal lesions. A dissociation between uniform motion and kinetic shape perception was also observed in patients with extensive white matter lesions (32) and in multiple sclerosis patients (33).

That the area postulated to be the MT homologue responds only weakly to kinetic contours is in agreement with physiological data showing that MT neurons respond less to kinetic gratings than to uniform random dot patterns (34, 35). MT neurons also respond weakly to stationary luminance-defined gratings (36), so that the effects of the Kin and Lum stimuli most likely canceled one another in our Kin – Lum subtractions. It has been suggested that in humans V1 contributes to motion segmentation (37). Since V1 neurons respond well to both luminance-defined and kinetic gratings, the signals in V1 may have been mutually canceled out in our Kin – Lum subtraction.

What the present results reveal is a cortical area that responds much more vigorously to kinetic gratings than to luminance-defined gratings and is not as well driven by uniformly moving random dot stimuli as the MT/V5 homologue. Two interpretations can be given to this kinetic area. Either it represents the preprocessing of motion for kinetic stimuli, implying that this preprocessing does not occur in MT/V5, or it represents the activity of neurons tuned to orientation of kinetic boundaries, which respond little to luminance-defined stimuli. In this latter case the preprocessing could still occur in MT/V5 and be relayed to the kinetic area. Inasmuch as this area does not respond well to luminance boundaries, such a projection would not really qualify as a reentrance (16). It is unclear whether this kinetic area has a counterpart in the monkey brain. An obvious candidate would be area V3, which also receives magnocellular input (38). However the homologue of V3 has been tentatively identified at higher levels (+16 min and above) by Watson *et al.* (20), and our own data support their view (29). In fact, an area in the monkey brain specialized for processing kinetic contours may have escaped experimenters who generally use uniformly moving random dot patterns to explore motion responses in monkey visual cortex.

We are indebted to Prof. R. Frackowiak for making available the SPM software and to Prof. I. Biederman for suggesting the counting paradigm. The technical help of C. Fransen, M. De Paep, S. Vleugels, L. Verhaegen, T. Degroot, M. Heroes, P. Kayenbergh, G. Meulemans, and Y. Celis is gratefully acknowledged. A. Rosier helped with the training of the subjects. We also thank R. Tootell, S. Raiguel, and V. Marcar for their comments on earlier versions of the manuscript. This work was supported by Grants 9.007.88, 3.0043.89, and 3.0095.92 from the Belgian National Research Council and by a grant from the Research Council of the Katholieke Universiteit Leuven. R. Vogels, B.D.B., and P.D. are research associates of the Belgian National Research Council; R. Vandenberghe is a research assistant of the National Research Council of Belgium.

1. Nakayama, K. (1985) *Vision Res.* **25**, 625–660.
2. Zeki, S. M. (1974) *J. Physiol. (London)* **236**, 549–573.
3. Van Essen, D. C., Maunsell, J. H. R. & Bixby, J. L. (1981) *J. Comp. Neurol.* **199**, 293–326.

4. Albright, T. D. (1984) *J. Neurophysiol.* **52**, 1106–1130.
5. Maunsell, J. H. R. & Van Essen, D. C. (1983) *J. Neurophysiol.* **49**, 1127–1146.
6. Lagae, L., Raiguel, S. & Orban, G. A. (1983) *J. Neurophysiol.* **69**, 19–39.
7. Newsome, W. T. & Paré, E. B. (1988) *J. Neurosci.* **8**, 2201–2211.
8. Vandenberghe, E., Saunders, R. C. & Orban, G. A. (1991) *Soc. Neurosci. Abstr.* **17**, 8.
9. Schiller, P. H. (1993) *Visual Neurosci.* **10**, 717–746.
10. Newsome, W. T., Wurtz, R. H., Dürsteler, M. R. & Mikami, A. (1985) *J. Neurosci.* **5**, 825–840.
11. Marcar, V. L. & Cowey, A. (1992) *Eur. J. Neurosci.* **4**, 1228–1238.
12. Marcar, V. L., Raiguel, S. E., Xiao, D., Maes, H. & Orban, G. A. (1991) *Soc. Neurosci. Abstr.* **17**, 525.
13. Sáros, Gy., Vogels, R. & Orban, G. A. (1993) *Science* **260**, 995–997.
14. Logothetis, K. & Charles, E. R. (1990) *Invest. Ophthalmol. Visual Sci. Suppl.* **31**, 444.
15. Marcar, V. L., Raiguel, S. E., Xiao, D., Maes, H. & Orban, G. A. (1992) *Soc. Neurosci. Abstr.* **18**, 1275.
16. Tononi, G., Sporns, O. & Edelman, G. M. (1992) *Cereb. Cortex* **2**, 310–335.
17. Ungerleider, L. G. & Mishkin, M. (1982) in *Analysis of Visual Behavior*, eds. Ingle, D. J., Goodale, M. A. & Mansfield, R. J. W. (MIT Press, Boston), pp. 549–586.
18. Fox, P. T., Mintun, M. A., Raichle, M. E., Miezin, F. M., Allman, J. M. & Van Essen, D. C. (1986) *Nature (London)* **323**, 806–809.
19. Zeki, S., Watson, J. D. G., Lueck, C. J., Friston, K. J., Kennard, C. & Frackowiak, R. S. J. (1991) *J. Neurosci.* **11**, 641–649.
20. Watson, J. D. G., Myers, R., Frackowiak, R. S. J., Hajnal, J. V., Woods, R. P., Mazziotta, J. C., Shipp, S. & Zeki, S. (1993) *Cereb. Cortex* **3**, 79–94.
21. Friston, K. J., Frith, C. D., Liddle, P. F., Dolan, R. J., Lammermsma, A. A. & Frackowiak, R. S. J. (1990) *J. Cereb. Blood Flow Metab.* **10**, 458–466.
22. Talairach, J. & Tournoux, P. (1988) *Co-Planar Stereotaxic Atlas of the Human Brain* (Thieme, New York), p. 122.
23. Friston, K. J., Frith, C. D., Liddle, P. F. & Frackowiak, R. S. J. (1991) *J. Cereb. Blood Flow Metab.* **11**, 690–699.
24. Dupont, P., Orban, G. A., Vogels, R., Schoups, A., Bormans, G., Nuyts, J. & Mortelmans, L. (1993) *Soc. Neurosci. Abstr.* **19**, 1501.
25. Dupont, P., Orban, G. A., Vogels, R., Bormans, G., Nuyts, J., Schiepers, C., De Roo, M. & Mortelmans, L. (1993) *Proc. Natl. Acad. Sci. USA* **90**, 10927–10931.
26. Orban, G. A., Duncan, J., Dupont, P., Ward, R., Bormans, G., De Roo, M. & Mortelmans, L. (1993) *Soc. Neurosci. Abstr.* **19**, 773.
27. Corbetta, M., Miezin, F. M., Dobmeyer, S., Shulman, G. L. & Petersen, S. E. (1991) *J. Neurosci.* **11**, 2383–2402.
28. Engel, S. A., Rumelhart, D. E., Wandell, B. A., Lee, A. T., Glover, G. H., Chichilnisky, E.-J. & Shadlen, M. N. (1994) *Nature (London)* **369**, 525.
29. Dupont, P., Orban, G. A., De Bruyn, B., Verbruggen, A. & Mortelmans, L. (1994) *J. Neurophysiol.* **72**, 1420–1424.
30. Chang, J. J. & Julesz, B. (1983) *Vision Res.* **23**, 639–646.
31. Vaina, L. M. (1989) *Biol. Cybern.* **61**, 347–359.
32. Regan, D., Giaschi, D., Sharpe, J. A. & Hong, X. H. (1992) *Vision Res.* **12**, 2198–2210.
33. Regan, D., Kothe, A. C. & Sharpe, J. A. (1991) *Brain* **114**, 1129–1155.
34. Snowden, R. J., Treue, S., Erickson, R. G. & Andersen, R. A. (1991) *J. Neurosci.* **11**, 2768–2785.
35. Marcar, V. L., Raiguel, S. E., Xiao, D., Maes, H. & Orban, G. A. (1991) *Soc. Neurosci. Abstr.* **17**, 525.
36. Albright, T. D. (1984) *J. Neurophysiol.* **52**, 1106–1130.
37. Lamme, V. A. F., Van Dijk, B. W. & Spekreijse, H. (1994) *Vision Res.* **34**, 721–735.
38. Felleman, D. J. & Van Essen, D. C. (1991) *Cereb. Cortex* **1**, 1–47.